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Right-Sided Air Embolism after Cardiopulmonary Bypass: Not Only a Left Side Problem

Silvina Longo¹
Martín Palacios¹
Germán Chaud²
Juan I. De Brahi^{1*}

1Department of Anesthesiology, Private University Hospital of Cordoba , Argentina
2Department of Cardiac-surgery, Private University Hospital of Cordoba , Argentina

Abstract

Transesophageal Echocardiography (TEE) is extremely important for detecting intracardiac air. We present two cases of air embolism identified in the pulmonary artery and right ventricle, after valvular surgery associated with right atrial opening. We analyzed the high sensitivity of TEE for an early diagnosis and the need for continuous monitoring during the Cardiopulmonary Bypass (CPB), because intracardiac air rapidly changes its location and appearance. We evaluated the incidence and procedures for eliminating intracardiac air. We emphasize that not only the left side of the heart should be analyzed, also to look for achieving a complete deaeration in the right heart cavities, especially if the right atrium was opened.

Keywords

TEE in Cardiac Surgery; Air Embolism in Open Heart Operation; Pulmonary artery embolism; Venous air embolism.

Introduction

The use of intraoperative Transesophageal Echocardiography (TEE) to detect intracardiac air is very useful and has been studied for many years, especially in cardiac surgery [1].

The echocardiogram clearly detects air bubbles as hyperechoic images because they are effective reflectors of ultrasound waves due to acoustic differences between air and blood. However most studies have reported left-sided heart air embolism and their potential detrimental effect, few studies have described, in cardiac surgery, severe complications that can result from significant air embolism detected in right-sided cardiac structure. We describe two cases in which air embolism was documented on the Right side.

Case Report

Case 1

A 72-year-old patient scheduled for mitral valve replacement due to severe insufficiency from a myxomatous valve with A2, A3 and P3 prolapse. A mechanical valve St Jude N-31 was placed through left atriotomy and coronary revascularization was performed. The CPB time was 166 minutes and aortic clamping 110 min. The weaning of CPB was performed without complications. Pulmonary artery systolic pressure was estimated from a modified bicava image, at two moments, before and after CPB (figures 1 and 2). In these images we can observe the increase of the pulmonary artery systolic pressure after CPB. In the evaluation with TEE no periprosthetic leaks were observed, the gradient was adequate for the valve and no abnormalities of myocardial contractility were observed. During the chest closure, the right atrium was injured and was repaired immediately. After that air was observed in the pulmonary artery. (video 1).

The patient was transferred to the Coronary Care Unit with 0.1 ug/kg/min of noradrenaline. Postoperative evolution was unfavorable, with persistent hypotension, hypoxemia and metabolic acidosis. The patient died 12 hours after de surgery.

Case 2

A 60-year-old patient with severe tricuspid insufficiency and right heart failure as a result of endocarditis (chemotherapy catheter-related that he had 10 years ago because of a multiple myeloma).

The tricuspid valve severely damaged was replaced by a mechanical mitral prosthesis N-33. A right atriotomy approach was used. The CPB time was 110 minutes and aortic clamping time was 73 minutes. In figure 3 we can observe the large dilatation of the right ventricle and the tricuspid annulus (ME 4 chamber view), and in Figure C we can see the intrahepatic venous dilatation (Transgastric view). As CPB was weaned, we observed, with TEE, a significant amount of air in the pulmonary artery, so active deaeration was implemented, reason why an additional time of CPB was needed until almost of the total

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*Corresponding author:

Juan I. De Brahi
Department of Anesthesiology
Private University Hospital of Cordoba
Cordoba, Argentina
E-mail: juanidebrahi@icloud.com

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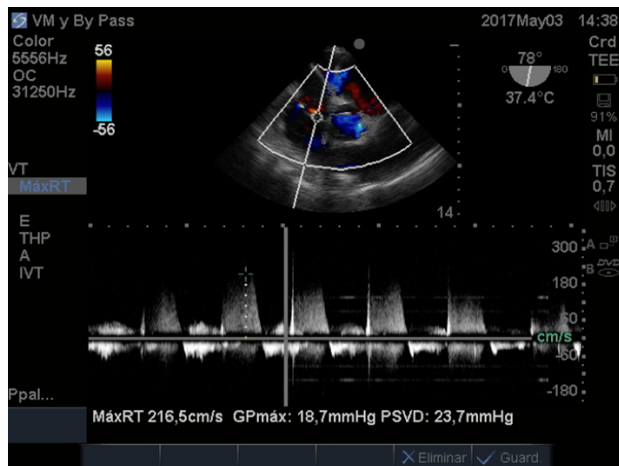


Figure 1: Modified bicava view: Continuous Doppler for evaluation of tricuspid reflux. Baseline pulmonary systolic pressure: 28.7 mmHg.

Right ventricular systolic pressure (RVSP) 23.7 mmHg+ 5 mmHg of central venous pressure (CVP) = 28.7 mmHg.

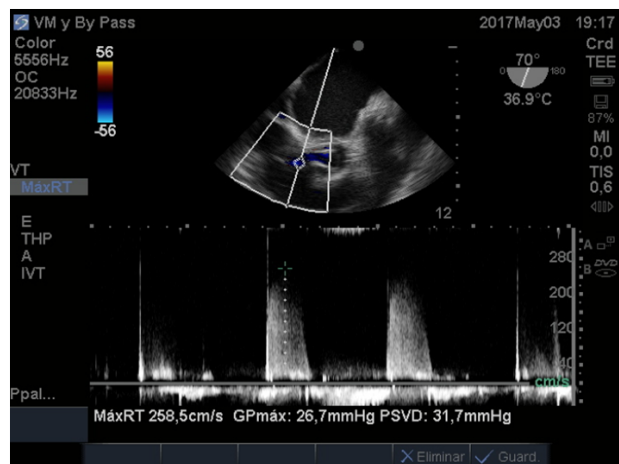


Figure 2: Modified bicava view: Continuous Doppler for evaluation of tricuspid reflux. Pulmonary systolic pressure (Post CPB) = 46.7 mmHg, Pulmonary hypertension.

RVSP = 31.7 mmHg+ 15 mmHg of CVP = 46,7 mmHg.



Figure 3: ME 4 chambersview – Dilatation of the tricuspid annulus, more than 4 cm.



Figure 4: Transgastric view – suprahepatics venous and inferior cava dilatation.

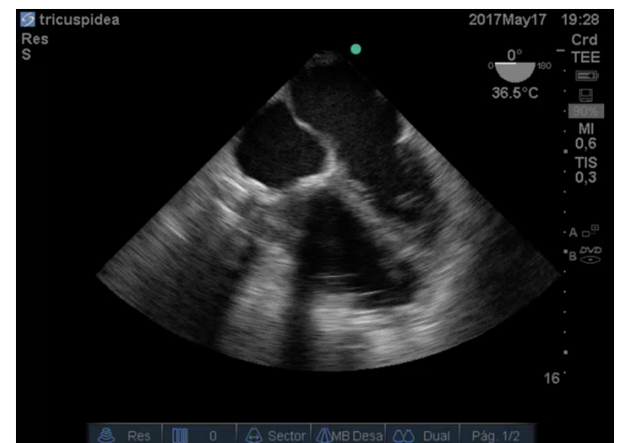


Figure 5: Mitral mechanical valve in tricuspid position.

air decreased. (video 2 pulmonary artery air conglomerate), (video 3 air-free pulmonary artery from upper esophageal window, aortic valve short axis view), (video 4: ME 4 chambers, mechanical valve in tricuspid position and air at the tip of Right ventricle).

The adequacy of the prosthetic valve was confirmed with TEE. For post-operative follow-up a Swan Ganz catheter was inserted in the CCU. The patient received 0.5ug/kg/ min noradrenaline and 0.375 ug/kg/min of milrinone. The pulmonary artery pressure was normal, the patient was extubated at 10 hours after surgery and after 10 days was discharged.

In both patients, interatrial communication through a patent foramen ovale was excluded.

At the time of weaning from CPB, air was observed in the right ventricle (Figure 6), and Figure 7 shows Echoes corresponding to conglomerates of air in the pulmonary artery. Before leaving CPB we could see absence of air after a good deaeration (Figure 8).

Discussion

At the end of the surgery, before leaving the CPB, the possible locations of trapped air must be investigated.

Due to differences in acoustic impedance between blood and particles, they will produce a white image on the echocardiogram. When the particles are air, they are called micro bubbles. It is important to discriminate between microbubbles and smoke (echoes of spontaneous contrast) seen in the heart cavities at different flow rates [5]. ETE showed that late episodes of microbubbles can be

seen in the pulmonary veins, suggesting that the air entered and was trapped in the vessels while the heart was open. Trapped air is not mobilized until normal blood flow is restored after CPB ends. The importance of air trapping in the pulmonary veins during heart operations were reported many years ago [6]. However, very little has been studied and reported.

Physiopathology

TEE is a very sensitive method to detect the presence of intracardiac air before leaving CPB. Widely studied and valued, air embolism in the left cavities can cause neurological damage, transient ventricular dysfunction and arrhythmias. A common situation of air

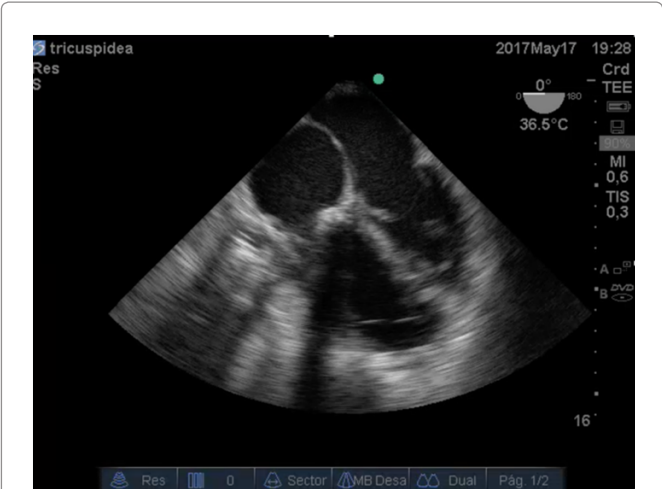
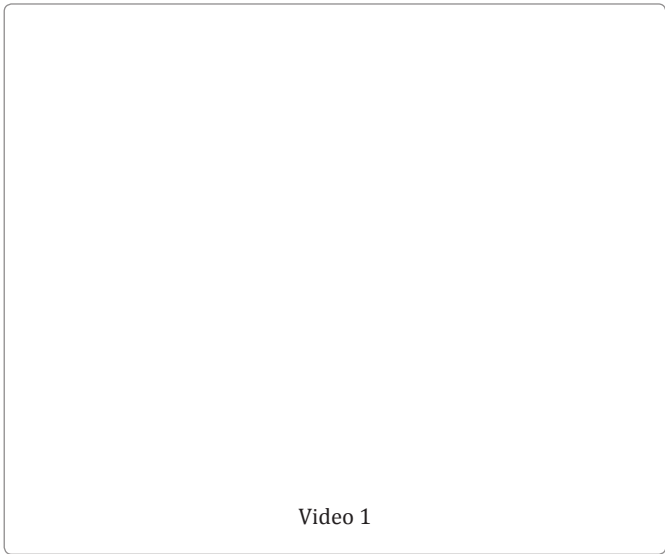


Figure 6: ME 4 chambers view, where you can see air in the apex of the Right Ventricle.



Video 1

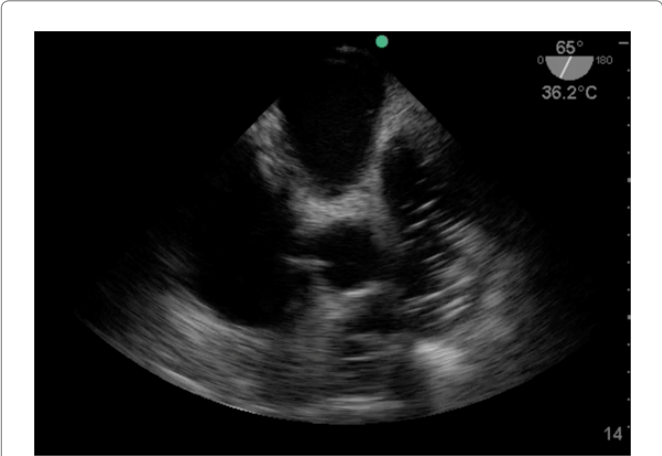
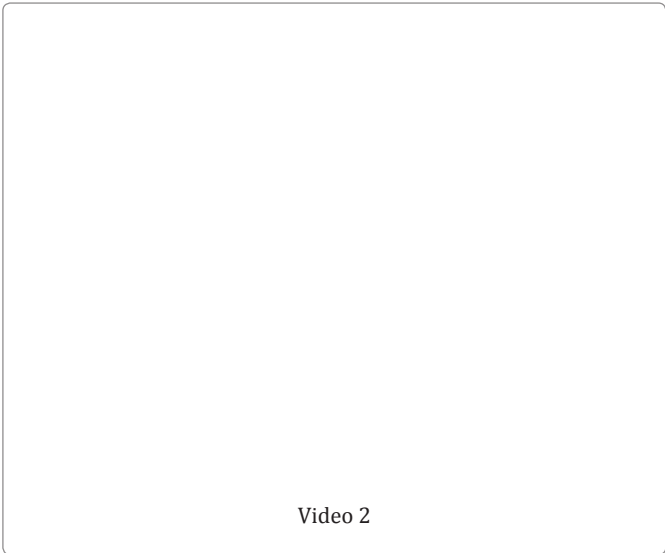


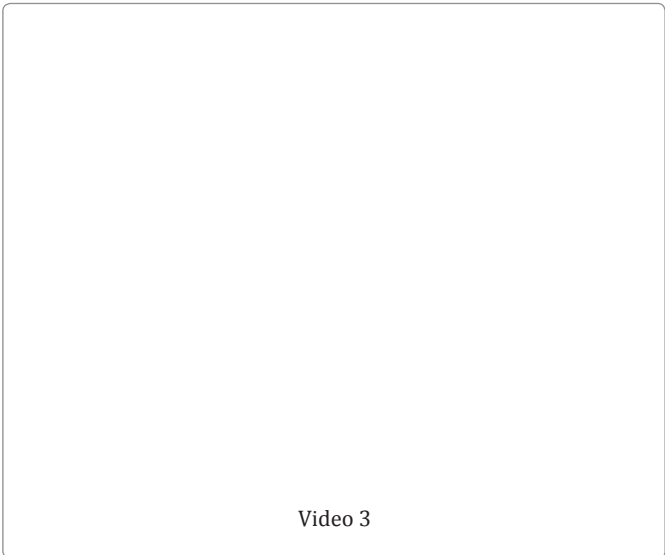
Figure 7: ME Aortic valve short axis, we can see several quantity of air in the Pulmonary artery.



Video 2



Figure 8: ME Aortic valve short axis where is no more air in the PA after the deaeration.



Video 3

Video 4

embolism, is the occurrence of transient right ventricular dysfunction at the end of CPB, secondary to airway obstruction of the right coronary artery, which is most frequently affected because of the anterior position of the right sinus of Valsalva. Other locations where air accumulation is frequent are the apex of the left ventricle, the left atrium and its appendix, and the pulmonary veins.

Air embolism is a rare but potentially fatal situation, and can result from a variety of procedures and scenarios.

It can be venous or arterial, depending on where the air enters into the circulatory system. Venous embolism occurs when air enters into a venous structure and travels through the right heart to the pulmonary circulation. The condition for the entry of air into the venous system is the presence of negative pressure in these vessels. The embolism can occur during placement of central venous catheters, based on a number of reported cases, ranging from 1 in 47 to 1 in 3000 [9,10]. It is most commonly associated with neurosurgical procedures. This is due to the location of the surgical incision that is generally superior to the heart at a distance that is greater than the central venous pressure. The seated position in posterior craniotomies is considered especially related to complications with the venous air procedure, estimated between 10% and 80% [11].

In both cases reported, the right atrium was opened, in the first patient accidentally, and in the second one to perform the tricuspid valve replacement. So we deduce that the air observed in the pulmonary artery has traveled rapidly from the atrium and the right ventricle to get trapped in this smaller vessel.

On the other side an arterial embolus occurs when air enters into an artery and travels until it gets trapped. A venous embolus always has the potential to become arterial if there is a connection between the two systems. For example, if a patient has a permeable foramen ovale, which is present in 30% of the general population, this can generate airflow from the right atrium to the left one if a pressure gradient occurs [7].

The incidence of massive air embolism in cardiac bypass is between 0.003% and 0.007% with 50% of adverse outcomes [8].

The physiological effects of air venous embolism are similar to those of pulmonary embolism of any etiology, as evidenced by:

1. Elevated pulmonary artery and right ventricle pressures.
2. Alteration of ventilation / perfusion ratio.
3. Intrapulmonary shunt.
4. Increased alveolar dead space.

The accumulation of air in the left ventricle stops the diastolic filling, and during systole air is pumped into the coronary arteries, interrupting coronary perfusion. Air in the vasculature results in hypoxemia, hypercapnia and acute changes in ventricular pressure, which can lead to right heart failure, decreased cardiac output, and

arrhythmia. This can be followed by systemic circulatory collapse, and even death.

The degree of physiological deterioration depends on air volume, air embolism rate, type of gas (ambient air, carbon dioxide or nitrous oxide), and the patient's position when embolism occurs.

The emboli not only cause a reduction in perfusion distal to the obstruction, also the damage results from an "inflammatory response" that the air bubble initiates. This inflammatory response and the changes it causes can result in pulmonary edema, bronchospasm and increased resistance of the respiratory tract.

Intracardiac air can be aspirated from the apex of the left ventricle and the aortic root with a needle. Vigorous massage can also be used to force the passage of air bubbles through the coronary arteries. Once the circulation is restored, the patient should be kept in the Trendelenburg position [12].

Complications

Damage to the central nervous system is always one of the most feared complications after cardiac operations [13].

There are several possible causes of these brain damage and air embolism is one of them.

Intracoronary air embolism is a complication reported in open heart surgery characterized by the appearance of hypotension, ST elevation, and fibrillation after the release of the aortic clamp or minutes after CPB. TEE can detect the presence of air in the coronary arteries and intraoperative myocardial ischemia due to segmental alterations of ventricular contractility [14].

When intracoronary embolism is detected, the treatment is symptomatic by increase the coronary perfusion pressure, and if necessary, reentry to CPB and add inotropic drugs to increase contractility [15,16].

If air embolism is suspected while the patient is on CPB, the perfusionist must stop the machine and cover all the catheters. The air must be out of the circuit and the patient placed in Trendelenburg position. TEE helps to locate the air and then it must be aspirated. Cooling of the patient, for neuroprotection purposes should be considered.

The study by Schafer et al was the first to analyze the persistence of intracardiac air and transit after venous embolism. The data show that after venous embolism, air appears in the right ventricle immediately after its appearance into the right atrium. In case of large venous embolism, air remains in the right ventricle for more than 12 min (animal study). In addition, air was detected for a considerably longer time in the right ventricle and pulmonary artery than in the right atrium. In the surgical setting, this gives an opportunity to aspirate the air from the right ventricle. A large catheter placed inside the artery may be an option to further improve air recovery¹⁷.

In our patients we believe that:

- In the first case we did not exhaust all the ways to achieve complete deaeration, perhaps a re-entry to CPB, punctured for air extraction or vigorous massage would have avoided the deleterious effects of air on the pulmonary circulation.
- In the second case we could notice it and we could ensure a complete deaeration with the help of the TEE, which contributed to the diagnosis and guided us in the therapeutics.

Conclusion

Air embolism is a life-threatening complication, which determines that prevention and early detection is an essential objective in Cardiac surgery with CPB.

With TEE, its detection can lead to treatment, requiring an extra time of extracorporeal circulation until complete deaeration of the heart.

Because intracardiac air rapidly changes its locations and appearances, continuous monitoring is important, especially at the end of the bypass.

At present it is a standard, to carry out a careful deaeration, not only attending to the left cavities, also obtaining its early detection in right cavities, especially the air that can enter through the venous or atrial circulation.

It is recommended a routine use of TEE during cardiac operations that leads to increased awareness of this problem and contributes to increased efforts to prevent or minimize damage to the Central Nervous System or lungs after cardiac surgery.

We must take into account that not only the left side of the heart should be analyzed, also as in the cases presented, look for and worry about achieving a complete deaeration in the right heart cavities, especially if the right atrium was opened. Air is still harmful to the brain and other organs such as the lungs, not only because of its direct action but also because of the inflammatory effects of a large embolism.

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